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MHP

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

DAVID APPLESTEIN, Individually and on Behalf of All Others Similarly Situated,

Plaintiff,

vs.

MEDIVATION, INC., DAVID T. HUNG, C. PATRICK MACHADO, LYNN SEELY and ROHAN PALEKAR,

Defendants.

No. CV 10 0998

CLASS ACTION

COMPLAINT FOR VIOLATION OF THE FEDERAL SECURITIES LAWS

DEMAND FOR JURY TRIAL

NATURE OF THE ACTION

1. This is a securities fraud class action on behalf of all purchasers of the common stock of Medivation, Inc. ("Medivation" or the "Company") between July 17, 2008 and March 2, 2010, inclusive (the "Class Period"), against Medivation and certain of its officers and directors for violations of the Securities Exchange Act of 1934 (the "1934 Act").

2. Medivation is a biopharmaceutical company with small molecule drugs in clinical development to treat three diseases: Alzheimer's disease, Huntington's disease and castration-resistant prostate cancer. During the Class Period, Medivation co-partnered with Pfizer, Inc. ("Pfizer") in the United Stated and overseas on the drug Dimebon (under the generic name latrepirdine), an experimental drug for Alzheimer's disease, which failed to benefit patients in an advanced study, causing millions of dollars in market capitalization losses.

SUMMARY AND OVERVIEW

3. During the Class Period, defendants made false and misleading statements regarding the Company's drug Dimebon. Specifically, throughout the Class Period, defendants violated the federal securities laws by disseminating false and misleading statements to the investing public about the effectiveness of Dimebon as a treatment for Alzheimer's disease, making it impossible for shareholders to gain a meaningful or realistic understanding of the drug's progress toward FDA approval and market success.

4. On March 3, 2010, before the market opened, defendants were forced to publicly disclose that Dimebon did not meet primary and secondary goals in a Phase 3 trial for patients with mild to moderate Alzheimer's disease. The trial further demonstrated that in some cases patients taking a placebo fared better than patients taking Dimebon.

5. As a result of this news, Medivation's stock plummeted $27.15 per share to close at $13.10 per share on March 3, 2010 – a one-day decline of 67% on volume of 45 million shares, nearly 72 times the average three-month daily volume.

6. As a result of defendants' false and misleading statements, Medivation stock traded at artificially inflated prices during the Class Period, reaching a high of $40.25 per share on March 2, 2010. The inflation in Medivation's stock price during the Class Period permitted the Company to

complete a secondary offering on May 27, 2009 of 3.1625 million shares of Medivation stock (including the 0ver-allotment) at $21 per share for proceeds of $62.4 million.

JURISDICTION AND VENUE

7. Jurisdiction is conferred by §27 of the 1934 Act. The claims asserted herein arise under §§10(b) and 20(a) of the 1934 Act and Rule 10b-5.

8. Venue is proper in this District pursuant to §27 of the 1934 Act. Many of the false and misleading statements were made in or issued from this District.

9. Medivation's executive offices are located in San Francisco, California, where the day-to-day operations of the Company are directed and managed.

THE PARTIES

10. Plaintiff David Applestein purchased Medivation common stock as described in the attached certification and was damaged thereby.

11. Defendant Medivation is a biopharmaceutical company. The Company focuses on the development of small molecule drugs for the treatment of Alzheimer's disease, Huntington's disease, and castration-resistant prostate cancer. Its product pipeline includes Dimebon, which was in a pivotal Phase 3 trial in patients with mild-to-moderate Alzheimer's disease, and a Phase 2 clinical trial in patients with mild-to-moderate Huntington's disease; and MDV3100, a Phase 1-2 clinical trial product for patients with castration-resistant prostate cancer. The Company has a collaboration agreement with Pfizer to develop and commercialize Dimebon for the treatment of Alzheimer's and Huntington's diseases.

12. Defendant David T. Hung ("Hung") co-founded Medivation. Hung is, and at relevant times was, President, Chief Executive Officer ("CEO") and a director of the Company. During the Class Period, Hung reaped over $3.5 million in insider trading proceeds by selling 150,000 shares of his Medivation stock at artificially inflated prices.

13. Defendant C. Patrick Machado ("Machado") co-founded Medivation. Machado is, and at relevant times was, Chief Financial Officer ("CFO") and Chief Business Officer of the Company. During the Class Period, while Medivation's stock price was inflated due to defendants'

false statements, Machado sold 160,000 shares of his Medivation stock for proceeds of nearly $4.5 million.

14. Defendant Lynn Seely ("Seely") is, and at relevant times was, Senior Vice President and Chief Medical Officer for the Company. During the Class Period, while Medivation's stock price was inflated due to defendants' false statements, Seely sold 90,000 shares of her Medivation stock for proceeds of nearly $2.2 million.

15. Defendant Rohan Palekar ("Palekar") joined the Company in January 2008. Palekar is, and at relevant times was, Chief Commercial Officer ("CCO") of the Company. During the Class Period, while Medivation's stock price was inflated due to defendants' false statements, Palekar sold 63,500 shares of his Medivation stock for proceeds of over $1.7 million.

16. The individuals named as defendants in ¶¶12-15 are referred to herein as the "Individual Defendants." The Individual Defendants, because of their positions with the Company, possessed the power and authority to control the contents of Medivation's quarterly reports, press releases and presentations to securities analysts, money and portfolio managers and institutional investors, *i.e.*, the market. Each defendant was provided with copies of the Company's reports and press releases alleged herein to be misleading prior to or shortly after their issuance and had the ability and opportunity to prevent their issuance or cause them to be corrected. Because of their positions and access to material non-public information available to them but not to the public, each of these defendants knew that the adverse facts specified herein had not been disclosed to and were being concealed from the public and that the positive representations which were being made were then materially false and misleading. The Individual Defendants are liable for the false statements pleaded herein, as those statements were each "group-published" information, the result of the collective actions of the Individual Defendants.

FRAUDULENT SCHEME AND COURSE OF BUSINESS

17. In addition to the above-described involvement, each Individual Defendant had knowledge of Medivation's problems and was motivated to conceal such problems. Defendant Hung, as CEO, was responsible for the press releases issued by the Company. Defendant Seely, as the Chief Medical Officer, was a key person responsible for the summary of the efficacy and

findings of clinical trials released to the public about Dimebon. Defendant Palekar, as CCO, was responsible for the sales and marketing of Dimebon. Each Individual Defendant sought to demonstrate that they could lead the Company successfully and commercialize the drug Dimebon.

18. Each defendant is liable for (i) making false statements, ***or*** (ii) failing to disclose adverse facts known to him or her about Medivation. Defendants' fraudulent scheme and course of business that operated as a fraud or deceit on purchasers of Medivation common stock was a success, as it (i) deceived the investing public regarding Medivation's prospects and business; (ii) artificially inflated the price of Medivation common stock; (iii) permitted defendants to complete a secondary offering of Medivation stock at $21 per share; (iv) allowed defendants Hung, Machado, Seely and Palekar to sell nearly $12 million worth of their own Medivation stock at artificially inflated prices; and (v) caused plaintiff and other members of the Class to purchase Medivation common stock at inflated prices.

CLASS ACTION ALLEGATIONS

19. Plaintiff brings this action as a class action pursuant to Rule 23 of the Federal Rules of Civil Procedure on behalf of all persons who purchased or otherwise acquired Medivation common stock during the Class Period (the "Class"). Excluded from the Class are defendants and their families, the officers and directors of the Company, at all relevant times, members of their immediate families and their legal representatives, heirs, successors or assigns and any entity in which defendants have or had a controlling interest.

20. The members of the Class are so numerous that joinder of all members is impracticable. The disposition of their claims in a class action will provide substantial benefits to the parties and the Court. Medivation has over 33.5 million shares of stock outstanding, owned by hundreds if not thousands of persons.

21. There is a well-defined community of interest in the questions of law and fact involved in this case. Questions of law and fact common to the members of the Class which predominate over questions which may affect individual Class members include:

(a) whether the 1934 Act was violated by defendants;

(b) whether defendants omitted and/or misrepresented material facts;

(c) whether defendants' statements omitted material facts necessary to make the statements made, in light of the circumstances under which they were made, not misleading;

(d) whether defendants knew or deliberately disregarded that their statements were false and misleading;

(e) whether the price of Medivation common stock was artificially inflated; and

(f) the extent of damage sustained by Class members and the appropriate measure of damages.

22. Plaintiff's claims are typical of those of the Class because plaintiff and the Class sustained damages from defendants' wrongful conduct.

23. Plaintiff will adequately protect the interests of the Class and has retained counsel who are experienced in class action securities litigation. Plaintiff has no interests which conflict with those of the Class.

BACKGROUND TO DEFENDANTS' SCHEME

24. Medivation is a biopharmaceutical company. The Company focuses on the development of small molecule drugs for the treatment of Alzheimer's disease, Huntington's disease, and castration-resistant prostate cancer. Its product pipeline includes Dimebon, which was in a pivotal Phase 3 trial in patients with mild-to-moderate Alzheimer's disease, and a Phase 2 clinical trial in patients with mild-to-moderate Huntington's disease; and MDV3100, a Phase 1-2 clinical trial product in patients with castration-resistant prostate cancer. The Company has a collaboration agreement with Pfizer to develop and commercialize Dimebon for the treatment of Alzheimer's and Huntington's diseases.

25. In 1983, Dimebon was approved in Russia as an over-the-counter oral antihistamine for the treatment of allergic rhinitis and allergic dermatitis. It was later taken off the market when better antihistamines were introduced with fewer side effects. It was never available in the United States.

26. In the early 1990's, research began into whether there could be a link between Dimebon and Alzheimer's disease. In 2001, in a Phase 1 clinical study involving 14 patients, Dimebon demonstrated efficacy on patients with mild to moderate Alzheimer's disease. Medivation

was founded in September 2003 by defendants Hung and Machado. The Company acquired the rights to Dimebon in October 2003. A Phase 2 study was approved by the Russian Ministry of Health in 2005. According to Medivation, the study involved 183 patients and was completed in 2007. The study data suggested that Dimebon significantly improved symptoms in patients with mild to moderate Alzheimer's disease.

27. In January 2008, Medivation won approval from the Food and Drug Administration ("FDA") to engage in a confirmatory Phase 3 trial with Dimebon based on the earlier research. Given that the Phase 2 trial was conducted in Russia, the FDA required the Company to perform a significant portion of the Phase 3 trial in the United States.

DEFENDANTS' FALSE AND MISLEADING STATEMENTS ISSUED DURING THE CLASS PERIOD

28. On July 17, 2008, Medivation issued a press release entitled "Medivation Announces Publication in The Lancet of Dimebon Pivotal Trial Results in Alzheimer's Disease – Dimebon Improved the Clinical Course of Alzheimer's Disease; Patients Experienced Statistically Significant Improvements in Memory and Thinking, Activities of Daily Living, Behavior and Overall Function," which stated in part:

> Medivation, Inc. today announced publication of the results of its first Alzheimer's disease pivotal clinical trial of the investigational drug Dimebon in the July 19, 2008 issue of The Lancet. ***In this double-blind, placebo-controlled trial, patients with mild-to-moderate Alzheimer's disease treated with Dimebon experienced statistically significant improvements compared to placebo in all the key aspects of the disease: memory and thinking, activities of daily living, behavior and overall function.***
>
> After both six months and a full year of treatment, Dimebon-treated patients were significantly better than placebo-treated patients on all key aspects of the disease. The benefit on the primary endpoint, the Alzheimer's Disease Assessment Scale-cognitive subscale (ADAS-cog) at six months, was highly significant ($p<0.0001$). Patients treated with Dimebon were also significantly improved at six months over baseline on all measures ($p=0.005$ on ADAS-cog). Dimebon's benefit over placebo continued to increase throughout the 12-month treatment period. At the end of 12 months, Dimebon-treated patients preserved their starting level of function on each measure of Alzheimer's disease.
>
> "In this study, Dimebon improved the clinical course of Alzheimer's disease, which is important given that the natural course is progressive deterioration over time," said Rachelle Doody, M.D., Ph.D., lead author and the Effie Marie Cain Chair in Alzheimer's Disease Research at the Alzheimer's Disease and Memory Disorders Center, Baylor College of Medicine in Houston. "There is a clear need for new treatments that can add value and enduring benefit to the treatment of Alzheimer's

disease. The results of this trial suggest that, if the findings are replicated, Dimebon could advance Alzheimer's treatment, offering more hope for patients and their caregivers."

Dimebon was well-tolerated throughout the trial. There was no difference between the Dimebon and placebo groups in the number of patients with adverse events, and the most common side effects seen were dry mouth (18 percent versus 1 percent for placebo) and depressed mood/depression (15 percent versus 5 percent for placebo). Importantly, fewer patients treated with Dimebon had serious adverse events than did patients on placebo at the end of the study (3 percent versus 12 percent; p=0.03).

Additional analyses of the Dimebon pivotal study data presented at recent medical conferences showed that Dimebon's impact extended to caregivers. Behavioral improvements in Dimebon-treated patients resulted in a significant decrease in caregiver distress at six months and at one year compared to the distress of caregivers of placebo-treated patients. Further, after six months, caregivers of Dimebon-treated patients saved approximately one hour per day assisting patients with activities of daily living compared to caregivers of placebo-treated patients.

"The magnitude, consistency and duration of the beneficial effects of Dimebon demonstrated in this trial are striking," said Paul Aisen, M.D., Director, Alzheimer's Disease Cooperative Study (ADCS) and Professor in the Department of Neurosciences, University of California, San Diego (UCSD). "In addition, the drug has been well-tolerated to date and, if the safety profile is replicated in the ongoing international trial, it will be a substantial advance for this patient population prone to drug side effects."

"We are pleased to see our first pivotal trial culminate with publication of its significant findings in such a prestigious journal," said David Hung, M.D., President and Chief Executive Officer of Medivation. ***"Currently available therapies treat the symptoms of Alzheimer's disease with only modest effect. The Dimebon study is the first study in which a drug has achieved statistically significant benefits of this breadth, size and duration in a one year, well-controlled trial.*** These data, coupled with our recently announced positive results in Huntington's disease, suggest that Dimebon could be a novel therapy for the treatment of neurodegenerative diseases. We look forward to the completion of our confirmatory pivotal Phase 3 study of Dimebon in Alzheimer's disease."

29. On this news, Medivation's stock increased from $14.94 per share to $18.61 per share – a one-day increase of $3.67 per share or 25%.

30. On July 30, 2008, Medivation issued a press release entitled "Medivation Presents Positive New Data on Dimebon's Long-Term Efficacy and Novel Mechanism of Action at the International Conference on Alzheimer's Disease," which stated in part:

Medivation, Inc. today announced new data showing that its investigational drug Dimebon continues to produce broad, clinically meaningful benefits in Alzheimer's disease patients after long-term dosing, and appears to operate through a novel mechanism of action. These data were presented today in a podium session and two poster sessions at the 2008 Alzheimer's Association International

Conference on Alzheimer's Disease (ICAD) in Chicago. The presentations are highlighted below.

Dimebon Preserves All Key Functions in Alzheimer's Patients for 18 Months in Open-Label Extension of First Pivotal Trial

New data from a six-month open-label extension of the 12-month placebo-controlled study of Dimebon in patients with mild-to-moderate Alzheimer's disease demonstrated that Dimebon continued to improve the clinical course of the disease. After 18 months of treatment, Dimebon preserved function in patients at or near their original levels upon entering the trial across all key aspects of Alzheimer's disease, specifically memory and thinking, behavior, activities of daily living and overall function. These results are noteworthy as untreated Alzheimer's patients progressively deteriorate over time in these areas. Dimebon remained well tolerated throughout the 18-month treatment period.

The open-label extension data were presented in a poster session by Jeffrey Cummings, M.D., Director of the Mary S. Easton Center for Alzheimer's Disease Research at UCLA. "To my knowledge, no other approved or investigational treatment has stabilized function across all facets of Alzheimer's disease for this length of time," said Dr. Cummings. "These data suggest that Dimebon may provide long-term benefits to Alzheimer's patients and provide further support for its potential as a promising therapeutic to treat this devastating disease."

Patients originally on placebo for 12 months who were then crossed over to Dimebon in the open-label extension phase stabilized across all key measures tested. Since these patients had declined over the previous 12 months while on placebo, they generally stabilized at a lower level of function than those treated with Dimebon for the full 18 months, suggesting a benefit of earlier treatment.

Dimebon Benefits Both Mild and Moderate Patients in 12-month Subgroup Analyses

New data from subgroup analyses by disease severity of the Dimebon double-blind placebo-controlled trial showed that Dimebon benefited both mild and moderate patients. In both mild and moderate patients, Dimebon treatment resulted in significant benefit on the study's primary endpoint, the Alzheimer's Disease Assessment Scale–cognition subscale, or ADAS-cog. The benefit in the moderate subpopulation was particularly robust, with a 9.7 point drug-placebo difference on the ADAS-cog ($p<0.0001$) after 12 months of treatment.

The subgroup analyses were presented in a separate poster presentation at ICAD 2008 by Rachelle Doody, M.D., Ph.D., the Effie Marie Cain Chair in Alzheimer's Disease Research at the Alzheimer's Disease and Memory Disorders Center, Baylor College of Medicine in Houston. "A nearly 10-point improvement over placebo in moderate patients on the ADAS-cog, a well-validated cognition scale in Alzheimer's disease, is unquestionably of clinical significance, especially in light of a clinical effect seen on the clinician's assessment of global function," said Dr. Doody. "If the results we saw for both the mild and moderate patients can be replicated, I believe that Dimebon will be an important advance in the treatment of Alzheimer's disease, regardless of stage."

Dimebon's Novel Mechanism of Action

In a podium presentation at ICAD 2008, Medivation presented new data on Dimebon's novel mitochondrial mechanism of action. Mitochondria generate energy

for cells and play important roles in mediating cell function and survival. Mitochondrial dysfunction has been linked in the published literature to both Alzheimer's and Huntington's diseases. Preclinical data presented showed that Dimebon improves mitochondrial function in the setting of cellular stress with very high potency. For example, Dimebon treatment improved mitochondrial function and increased the number of surviving cells after treatment with a cell toxin known as ionomycin in a dose-dependent fashion. The effect of Dimebon to improve mitochondrial dysfunction has been confirmed in the independent laboratory of Maria Ankarcrona, Ph.D., Associate Professor at the Karolinska Institutet in Sweden.

"All of the approved Alzheimer's disease drugs operate by one of two mechanisms – cholinesterase inhibition or NMDA-receptor antagonism," noted Bengt Winblad, M.D., Ph.D., Head of the Karolinska Institutet's Alzheimer's Disease Research Center. "The body of preclinical and clinical data generated thus far convinces me that Dimebon is exerting its effects through a different mechanism. The data presented today support the hypothesis that Dimebon improves mitochondrial dysfunction. This is a novel mechanism that may, in part, explain the clinical benefits seen in Alzheimer's patients treated with Dimebon."

About the Pivotal Study Dimebon's first pivotal Alzheimer's trial was a randomized, double-blind, placebo-controlled study of 183 patients with mild to moderate Alzheimer's disease. In this study, patients treated with Dimebon experienced statistically significant improvements compared to placebo in all the key aspects of the disease: memory and thinking, activities of daily living, behavior and overall function – after both six months and a full year of treatment. Dimebon's benefit over placebo continued to increase throughout the 12-month treatment period. At the end of 12 months, Dimebon-treated patients preserved their starting level of function on each measure of Alzheimer's disease. Results of the pivotal study were published in the July 19, 2008 issue of The Lancet.

Earlier this year, the U.S. Food and Drug Administration (FDA) informed Medivation that this study can be used as one of the pivotal studies required to support the approval of Dimebon to treat mild-to-moderate Alzheimer's disease, as long as a significant proportion of the sites in the confirmatory Phase 3 trial are located in the United States. The Company recently began a confirmatory pivotal Phase 3 trial of Dimebon in Alzheimer's disease known as the CONNECTION study.

31. On August 11, 2008, Medivation reported its second quarter 2008 financial results, in a release which stated in part:

Medivation, Inc. today reported on its corporate progress and financial results for the quarter ended June 30, 2008.

"Based on the significant findings of the Dimebon 12-month pivotal trial in Alzheimer's disease recently published in The Lancet, as well as the promising results from our Phase 2 study in Huntington's disease announced last month, we believe Dimebon is among the most promising drug candidates being investigated today to treat patients with debilitating, and ultimately fatal, neurodegenerative diseases," said David Hung, M.D., president and chief executive officer of Medivation. "We are making excellent progress opening U.S. sites and enrolling patients in our confirmatory Phase 3 trial of Dimebon in Alzheimer's disease, and remain on target to complete the study in time to file for U.S. marketing approval for Alzheimer's disease in 2010. In addition, we continue to increase the dose and enroll

patients in our ongoing Phase 1-2 study of MDV3100 for castration-resistant prostate cancer. We remain on track for completing that study later this year, after which we intend to seek FDA approval to enter Phase 3 in 2009."

Second Quarter Highlights and Recent Accomplishments

Alzheimer's Disease

- Initiated dosing of patients in a second pivotal Phase 3 trial of the investigational drug Dimebon in patients with mild-to-moderate Alzheimer's disease. This international, double-blind, placebo-controlled safety and efficacy study of oral Dimebon, known as the CONNECTION study, will enroll approximately 525 patients at 60 to 80 clinical sites in the U.S., Europe and South America.

- Published results of the first pivotal clinical trial of Dimebon in the July 19, 2008 issue of The Lancet. The article highlighted that patients with mild-to-moderate Alzheimer's disease treated with Dimebon experienced statistically significant improvements compared to placebo on all of the key aspects of the disease – memory and thinking, activities of daily living, behavior and overall function – over a 12-month period.

- Presented new Dimebon data at three presentations at the 2008 Alzheimer's Association International Conference on Alzheimer's Disease (ICAD):

  - Presented results from a six-month, open-label extension of the 12-month placebo-controlled study showing that Dimebon continued to improve the clinical course of the disease. After 18 months of treatment, Dimebon preserved function in patients at or near their original levels upon entering the trial across all key aspects of Alzheimer's disease. Dimebon remained well tolerated throughout the 18-month treatment period.

  - Presented new 12-month data from subgroup analyses by disease severity of the first pivotal trial showing that Dimebon benefited both mild and moderate patients, resulting in significant benefit on the study's primary endpoint, the Alzheimer's Disease Assessment Scale-cognitive subscale (ADAS-cog). The drug-placebo difference in moderate patients was 9.7 ADAS-cog points after 12 months of Dimebon treatment.

  - Presented new preclinical data at a podium presentation on Dimebon's novel mechanism of action, showing that Dimebon improves mitochondrial function in the setting of cellular stress with very high potency. Mitochondria, which generate energy for cells and play important roles in mediating cell function and survival, have been associated with both Alzheimer's and Huntington's diseases in the published literature.

- Successfully completed a thorough QTc cardiac safety study of Dimebon. In this study, Dimebon was well tolerated and did not produce any cardiac safety issues. The U.S. Food and Drug Administration requires thorough QTc studies for all new drugs undergoing regulatory approval.

32. On September 3, 2008, Medivation issued a press release entitled "Pfizer and Medivation Enter into Global Agreement to Co-Develop and Market Dimebon for the Treatment of Alzheimer's and Huntington's Diseases," which stated in part:

> Pfizer Inc. and Medivation, Inc. announced today that they have entered into an agreement to develop and commercialize Dimebon, Medivation's investigational drug for treatment of Alzheimer's disease and Huntington's disease. Dimebon currently is being evaluated in an international, confirmatory Phase III trial in patients with mild-to-moderate Alzheimer's disease (www.connectionstudy.com).
>
> Under the terms of the agreement, Medivation will receive an up-front cash payment of $225 million. Medivation also is eligible to receive payments of up to $500 million upon the attainment of development and regulatory milestones plus additional undisclosed commercial milestone payments. Medivation and Pfizer will collaborate on the Phase III program in Alzheimer's disease, Huntington's disease development and regulatory filings in the United States. The companies will share all U.S. development and commercialization expenses along with U.S. profits/losses on a 60 percent/40 percent basis, with Pfizer assuming the larger share of both expenses and profit/losses. In addition, Medivation will co-promote Dimebon to specialty physicians in the U.S.
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> Pfizer will have responsibility for development, regulatory and commercialization outside the U.S. and will pay Medivation tiered royalties on commercial sales outside of the U.S. The agreement is subject to approval under the Hart-Scott-Rodino Antitrust Improvements Act of 1976. J.P. Morgan served as financial advisor, and Cooley Godward Kronish LLP served as legal advisor, to Medivation on this transaction.
>
> Alzheimer's disease leads to the death of brain cells and the loss of nerve connections in areas of the brain that govern memory, thinking and behavior. Alzheimer's disease gradually destroys a person's memory and ability to learn, reason, make judgments, communicate and carry-out daily activities. No currently marketed Alzheimer's disease drug appears to stop brain cell death and prevent or restore lost nerve connections.
>
> Dimebon is an orally-available, small molecule that has been shown to inhibit brain cell death in preclinical models relevant to Alzheimer's disease and Huntington's disease, making it a potential treatment for these and other neurodegenerative conditions. Based on preclinical data generated to date, Dimebon appears to improve the function of mitochondria, the energy generators in cells that play a vital role in governing brain cell health, growth and overall function. Dimebon also has been shown to stimulate the outgrowth of nerves from brain cells, or neurites, a process that is believed to play an important role in restoring or generating new brain cell connections.
>
> "With more than 18 million people worldwide suffering from the debilitating and ultimately fatal effects of Alzheimer's disease, Pfizer has made this devastating illness one of our highest priorities," said Dr. Martin Mackay, president, Pfizer Global Research and Development. "We are working to develop new medicines that improve memory and halt or significantly slow the disease's progression. We look forward to collaborating with Medivation to bring Dimebon to patients as rapidly as possible."

"After a rigorous process that garnered substantial interest, we believe that Pfizer is the ideal partner, sharing our vision for Dimebon and capable of maximizing its potential globally," said Dr. David Hung, president and chief executive officer of Medivation. "As one of the leaders in Alzheimer's disease, Pfizer is an optimal partner because of its extensive experience developing new medicines; its marketing and commercialization track record; and, its significant global capability to effectively reach primary care physicians, who today prescribe the vast majority of Alzheimer's disease medications in the U.S."

33. On November 10, 2008, Medivation reported its third quarter 2008 financial results in a release which stated in part:

Medivation, Inc. today reported on its corporate progress and financial results for the quarter ended September 30, 2008.

"The third quarter represented another quarter of significant achievement and progress and was capped with the signing of our partnering agreement with Pfizer for Dimebon. This collaboration not only gives us access to a world-class partner capable of maximizing global commercialization, but also provides significant funding allowing us to invest in all of our clinical programs and actively pursue other drug candidates," said David Hung, M.D., president and chief executive officer of Medivation. "We are working with Pfizer on an extensive program to support a broad label for Dimebon in Alzheimer's disease beyond our original plan to pursue the treatment of mild-to-moderate Alzheimer's, and to achieve comprehensive and expeditious regulatory submissions and market acceptance. Accordingly, together we intend to expand development of Dimebon to include new Phase 3 trials in addition to the CONNECTION study. We expect to begin the new trials in 2009 and to file a New Drug Application (NDA) for a broader Alzheimer's disease label in 2011."

Corporate Update

Dimebon: Drug candidate to treat Alzheimer's and Huntington's diseases

- Entered into an agreement with Pfizer Inc. to jointly develop and commercialize Dimebon for the treatment of Alzheimer's and Huntington's diseases. Under the terms of the agreement, Medivation has received an up-front cash payment of $225 million and is eligible to receive payments of up to $500 million upon the attainment of development and regulatory milestones, plus additional undisclosed commercial milestone payments.

- Enrollment in CONNECTION, our confirmatory Phase 3 trial in mild-to-moderate Alzheimer's disease, continues on track. All 30 of our U.S. sites have been opened, and we expect the majority of our ex-U.S. sites to be opened by the end of November. We expect to complete enrollment of this trial in 2009.

- Completed a randomized, double-blind safety and tolerability study of combination therapy with Dimebon and donepezil (Aricept(R)) in patients with Alzheimer's disease, which found the combination to be well tolerated with no serious adverse events.

- Plan to initiate new Phase 3 studies in 2009 to seek further differentiation of Dimebon to include moderate-to-severe

Alzheimer's disease, adjunctive use with cholinesterase inhibitors, and twelve-month efficacy.

– Received a Corporate Achievement Award from the Huntington's Disease Society of America (HDSA) for exemplifying leadership in the fight against Huntington's disease and other neurodegenerative diseases.

– Plan to initiate the next Huntington's disease efficacy study in 2009.

34. On December 9, 2008, Medivation issued a press release entitled "Medivation Presents New Data on Dimebon's Novel Mechanism of Action – Dimebon Shown to Impact Two Key Aspects of Brain Cell Function," which stated in part:

> Medivation, Inc. presented new data that provide additional evidence that Dimebon, its lead product candidate in development to treat Alzheimer's and Huntington's diseases, potentially operates via a novel mitochondrial mechanism of action. In preclinical studies, Dimebon was shown to impact two key aspects of brain cell function: it promoted neurite outgrowth and it preserved mitochondrial function after brain cells were challenged with beta amyloid, a toxic substance often associated with Alzheimer's disease and the loss of brain cells.
>
> "In experiments in which brain cells were exposed to different toxins, including beta amyloid, Dimebon was shown to stabilize mitochondrial function, a vital element of neuron function and survival," said Andrew Protter, Ph.D., vice president, preclinical development for Medivation. ***"These findings suggest that Dimebon may have benefits on slowing the progression of Alzheimer's disease by preserving mitochondrial function. This potential novel mechanism may help explain the clinical benefits seen to date in Alzheimer's patients treated with Dimebon."***
>
> Dr. Protter presented the new data in an oral presentation, entitled "Dimebon Induces Neurite Outgrowth and Stabilization in the Setting of Cell Stress," at Cold Spring Harbor Laboratory's "Neurodegenerative Diseases: Biology & Therapeutics" meeting.
>
> Mitochondria and Cell Function
>
> Mitochondria generate energy for cells and play important roles in mediating cell function and survival. Improved mitochondrial function has been correlated with increased synapse formation. Autopsy studies of brains from patients with Alzheimer's disease suggest that mitochondrial damage and synapse dysfunction are early cellular events in Alzheimer's disease development and progression. Similarly, mitochondrial dysfunction has been linked in the published literature to the progression of Huntington's disease.
>
> Preclinical Study Results
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> As synapse formation is dependent on mitochondrial function and synapse loss is a major characteristic observed in the brain tissue of individuals with Alzheimer's disease, researchers evaluated the effects of Dimebon on neurite outgrowth, an important aspect of synapse formation.

Results of the study showed that Dimebon induced a statistically significant increase in neurite outgrowth from cortical, hippocampal and spinal cord neurons. Dimebon's potent effect on neurite outgrowth was seen at low concentrations and was comparable to that achieved with maximally effective concentrations of a potent growth factor (Brain Derived Neurotrophic Factor). Study results also showed that Dimebon reduced mitochondrial impairment in the setting of cellular stress. Specifically, Dimebon treatment mitigated mitochondrial impairment induced by beta amyloid.

Dimebon's effect on improving mitochondrial dysfunction has been confirmed previously in the independent laboratory of Maria Ankarcrona, Ph.D., associate professor at the Karolinska Institutet in Sweden. Additional data about Dimebon's potential novel mechanism of action were presented in November at the Society for Neuroscience's "Neuroscience 2008" conference in Washington, D.C.

Preclinical data also have been presented that suggest that Dimebon works through a different mechanism of action than other drugs that focus on targets implicated in cognition and memory loss, such as cholinesterase inhibition. In these experiments, Dimebon was shown to be a weak cholinesterase inhibitor, and additional data from binding assays showed that Dimebon did not have strong affinity to other standard targets. This suggests that Dimebon's potential novel mitochondrial mechanism of action may account for the clinical benefit observed in the Dimebon Alzheimer's and Huntington's disease clinical trials completed to date.

35. On March 16, 2009, Medivation reported its fourth quarter and year-end 2008 financial results in a release which stated in part:

Medivation, Inc. today reported on its corporate progress and financial results for the fourth quarter and year ended December 31, 2008.

"We had a very productive 2008 and are looking forward to an equally exciting 2009. We have made excellent progress across our pipeline and are on track to be in Phase 3 testing in all of our programs in 2009 – Dimebon in both Alzheimer's and Huntington's diseases and MDV3100 for castration-resistant prostate cancer," said David Hung, M.D., president and chief executive officer of Medivation. "We and Pfizer are jointly executing a comprehensive Phase 3 development program for Dimebon in Alzheimer's disease and are already well on our way with the ongoing CONNECTION study, the recent initiation of a large safety trial designed to support a potential earlier NDA filing date, and the soon-to-be-initiated CONCERT study of Dimebon in combination with Aricept. In addition, with Pfizer, we had a successful end-of-Phase 2 meeting with the FDA for Dimebon in Huntington's disease."

Corporate Update

Dimebon

Alzheimer's Disease:

- On track with patient enrollment in CONNECTION, a confirmatory Phase 3 trial in mild-to-moderate patients, allowing for expected completion of enrollment in 2009. We have 66 open sites in the U.S., Europe and South America.

- Initiated a placebo-controlled Phase 3 safety study in 750 Alzheimer's disease patients on a variety of background antidementia drugs. The purpose of the safety study is to generate a sufficient safety database to give us the option for an earlier-than-planned filing of our initial marketing application should we and Pfizer elect to pursue that option.

- Plan to initiate enrollment next month in our Phase 3 CONCERT trial, a 12-month safety and efficacy study evaluating Dimebon in combination with donepezil (Aricept(R)) in approximately 1,000 patients with mild-to-moderate Alzheimer's disease.

- Plan to initiate two additional Phase 3 studies in 2009 that will evaluate Dimebon in a total of approximately 1,100 patients with moderate-to-severe Alzheimer's disease.

* * *

Mechanism of Action (MOA)

- Presented new data at various medical conferences, including the recent AD/PD conference, that provide additional evidence that Dimebon potentially stabilizes and improves mitochondrial function in a way that prevents neuron death and dysfunction, a mechanism thought to be distinct from currently available Alzheimer's disease medications.

- At AD/PD, we presented new research from the Karolinska Institutet, which quantified the impact of Dimebon on mitochondrial function. Responses were seen on mitochondrial function at low concentrations of Dimebon across multiple experiments, and Dimebon showed potent mitochondrial responses in both stressed and normal cells.

36. On April 15, 2009, Medivation issued a press release entitled "Pfizer and Medivation Initiate Phase 3 Trial of Dimebon Added to Donepezil in Patients with Alzheimer's Disease – New 12-month study broadens Phase 3 clinical program to further evaluate the benefits of Dimebon in Alzheimer's Disease," which stated in part:

> Pfizer and Medivation, Inc. today announced the initiation of a 12-month, Phase 3 clinical trial of the investigational drug Dimebon. The study, known as CONCERT, is designed to evaluate the safety and efficacy of Dimebon when added to ongoing treatment with donepezil HCl tablets, the leading Alzheimer's disease (AD) medication worldwide, in patients with mild-to-moderate AD.
>
> The CONCERT study is part of a broad, Phase 3 clinical development program for Dimebon. The study builds on data from a small-scale safety and tolerability trial of Dimebon added to donepezil, which found the combination to be well tolerated. CONCERT is designed to complement previous and ongoing studies by further evaluating the efficacy of Dimebon. The Phase 3 program also includes the confirmatory 6-month CONNECTION study, which is designed to evaluate the safety and efficacy of Dimebon monotherapy in patients with mild-to-moderate AD and builds on results of the first pivotal trial of Dimebon in AD.

"Due to the complexity of Alzheimer's disease, the condition often requires combination treatment to help relieve symptoms and slow disease progression," said Bengt Winblad, professor of geriatrics, Karolinska Institute. "The CONCERT trial will explore the potential additive effects of Dimebon to ongoing donepezil therapy, two drugs thought to have different mechanisms of action. We believe this trial may serve to demonstrate the potential of Dimebon in AD."

Dimebon is an investigational compound currently in Phase 3 development for the treatment of Alzheimer's disease (AD) and in clinical development for Huntington's disease (HD). In preclinical models of AD and HD explored thus far, Dimebon has been shown to inhibit brain cell death, potentially by stabilizing and improving mitochondrial function in a way that prevents neuron death and dysfunction. The Dimebon mechanism is thought to be distinct from currently available AD medications.

Design of the CONCERT Study

The international, randomized, double-blind, placebo-controlled study will enroll approximately 1,050 patients with mild-to-moderate AD at approximately 100 sites in the United States, Australia, New Zealand and Western Europe. Patients on a stable dose of donepezil will be randomized to one of three treatment groups: Dimebon 20 mg three times per day, Dimebon 5 mg three times per day or placebo. Patients must be on treatment with donepezil for at least six months and at a stable dose of 10 mg daily for at least four months prior to enrollment in the study.

37. On May 11, 2009, Medivation reported its first quarter 2009 financial results in a release which stated in part:

Medivation, Inc. today reported on its corporate progress and financial results for the first quarter ended March 31, 2009.

"***We continue to make significant progress with both of our product candidates – Dimebon in patients with Alzheimer's and Huntington's diseases*** and MDV3100 in patients with prostate cancer. Having received written permission from the FDA to initiate a pivotal Phase 3 trial of MDV3100 in castration-resistant prostate cancer, we are on track to be in Phase 3 testing in all of our clinical programs this year," said David Hung, M.D., president and chief executive officer of Medivation. "We expect to achieve a significant milestone in June - completion of enrollment in our six-month, confirmatory, pivotal Phase 3 CONNECTION trial in mild-to-moderate Alzheimer's disease. And as part of our plan to support a broad and differentiated label for Dimebon in Alzheimer's disease, we are pleased to have initiated the Phase 3 CONCERT trial of Dimebon in combination with donepezil (Aricept(R)), and intend to begin two additional Phase 3 trials in moderate-to-severe Alzheimer's disease patients this year."

Recent Highlights and Accomplishments

Dimebon

- Expect to complete patient enrollment in CONNECTION, a confirmatory, pivotal Phase 3 trial in patients with mild-to-moderate Alzheimer's disease, in June.

- Initiated the Phase 3 CONCERT trial in patients with mild-to-moderate Alzheimer's disease; the 12-month clinical trial is designed to evaluate the efficacy of Dimebon when added to ongoing treatment with donepezil (Aricept(R)), the leading Alzheimer's disease medication worldwide, and builds on data from a small-scale safety and tolerability trial of Dimebon added to donepezil, which found the combination to be well tolerated.

- Completed a multicenter, randomized, double-blind, placebo-controlled Phase 1 study to evaluate the safety and tolerability of Dimebon given to Alzheimer's disease patients who currently are on a stable dose and regimen of memantine (Namenda(R)) or memantine plus donepezil. The study showed that these combinations were well tolerated.

- In addition to the CONNECTION and CONCERT trials and a Phase 3 safety study already underway, we and Pfizer plan to initiate two additional Phase 3 studies in 2009 that will evaluate Dimebon in a total of approximately 1,100 patients with moderate-to-severe Alzheimer's disease.

- Expect to initiate a Phase 3 trial this year to evaluate Dimebon's potential benefits on cognition in patients with mild-to-moderate Huntington's disease.

- Presented posters featuring Dimebon at the 61st American Academy of Neurology Annual Meeting in Seattle on April 29 and 30, including a poster entitled "Estimating Disease-Modifying Effects Using a Staggered Start Approach and a Natural History Staggered Start (NHSS) Approach: Preliminary Results from a 12-Month Study of Dimebon and a 6-Month Open-Label Period."

38. On May 27, 2009, Medivation announced the pricing of a secondary public offering, selling 2.75 million shares of its common stock at $21 per share, additionally granting the underwriters a 30-day option to purchase up to an additional 412,500 shares of common stock to cover over-allotments, generating $62.4 million in proceeds for the Company. The Prospectus for the offering stated in part:

Our Dimebon program

Potential neuroprotective activity

In preclinical experiments, Dimebon demonstrated neuroprotective activity in models relevant to Alzheimer's disease and Huntington's disease. The ß-amyloid protein is a known neurotoxin that is widely believed to contribute to the neurofibrillary tangles and plaques that characterize Alzheimer's disease. When neurons are exposed to the ß-amyloid protein in vitro, a significant portion of them die. Dimebon has been shown to inhibit this ß-amyloid induced neuron death in vitro. In addition, in a transgenic *Drosophila* (fruit fly) model of Huntington's disease, Dimebon has been shown to protect photoreceptor neurons against death induced by

the human gene encoding the huntingtin protein, an abnormal protein widely believed to cause Huntington's disease.

Mechanism of action

We believe that Dimebon is exerting its activity through a novel mechanism of action involving enhancement of mitochondrial function. Mitochondria are intracellular structures that are responsible for generating energy within all cells and play important roles in mediating brain cell function and survival. Mitochondrial dysfunction has been linked in the published literature to both Alzheimer's and Huntington's diseases. In addition, autopsy studies of brains from patients with Alzheimer's disease suggest that mitochondrial damage and synapse dysfunction are early cellular events in Alzheimer's disease development and progression.

In July 2008, we presented new preclinical data on Dimebon's novel mitochondrial mechanism of action. These data showed that Dimebon improves mitochondrial function in the setting of cellular stress with very high potency. For example, Dimebon treatment improved mitochondrial function and increased the number of surviving cells in a dose-dependent fashion after treatment with a cell toxin known as ionomycin.

In December 2008, we announced preclinical data that demonstrated that Dimebon impacted two key aspects of brain cell function: promotion of neurite outgrowth and preservation of mitochondrial function after brain cells were challenged with beta amyloid, a toxic substance often associated with Alzheimer's disease and the loss of brain cells. Results of the study showed that Dimebon induced a statistically significant increase in neurite outgrowth from cortical, hippocampal and spinal cord neurons. Dimebon's potent effect on neurite outgrowth was seen at low concentrations and was comparable to that achieved with maximally effective concentrations of a potent naturally occurring protein that is known to enhance brain cell function (Brain Derived Neurotrophic Factor). Study results also showed that Dimebon reduced mitochondrial impairment in the setting of cellular stress. Specifically, Dimebon treatment mitigated mitochondrial impairment induced by beta amyloid.

In addition, we believe based on preclinical data that Dimebon works through a different mechanism of action than other drugs that focus on targets implicated in cognition and memory loss, such as cholinesterase inhibition. In these preclinical experiments, Dimebon was shown to be a weak cholinesterase inhibitor, and additional data from binding assays showed that Dimebon did not have strong affinity to other standard targets. This suggests that Dimebon's potential novel mitochondrial mechanism of action may account for the clinical benefit observed in the Dimebon Alzheimer's and Huntington's disease clinical trials completed to date.

39. The offering was successful and the overallotment was fully subscribed.

40. On June 11, 2009, Medivation issued a press release entitled "Medivation Completes Enrollment in Confirmatory, Pivotal Phase 3 'CONNECTION' Trial of Dimebon in Patients With Alzheimer's Disease," which stated in part:

> Medivation, Inc. today announced the completion of patient enrollment in the CONNECTION study, a six-month, confirmatory, pivotal Phase 3 trial of the investigational drug dimebon in patients with mild-to-moderate Alzheimer's disease.

The international, double-blind, placebo-controlled, pivotal Phase 3 study enrolled 598 patients, exceeding the enrollment target of 525 patients. More than 40 percent of the patients enrolled were in the United States. The six-month study is evaluating the effect of dimebon on the Alzheimer's Disease Assessment Scale-cognitive subscale (ADAS-cog) and the Clinician's Interview-Based Impression of Change plus caregiver interview (CIBIC-plus) – the two endpoints have historically been accepted by the U.S. Food and Drug Administration (FDA) to support registration of currently approved drugs for mild-to-moderate Alzheimer's disease.

"Completion of patient enrollment in this second pivotal trial moves us closer to our goal of submitting a marketing application to the FDA and bringing dimebon to market for the many Alzheimer's patients suffering from this devastating disease," said Lynn Seely, M.D., chief medical officer of Medivation. "We are gratified by the strong interest in this trial as indicated by our exceeding the enrollment goal. Together with our partner Pfizer, we are executing a comprehensive clinical plan to support an NDA filing, currently targeted for 2011, with a broad and differentiated label for dimebon in Alzheimer's disease. We are also conducting a Phase 3 safety study, which will provide us and Pfizer the opportunity to seek marketing approval earlier if results of the CONNECTION study confirm our previously completed first pivotal study, which was published in the Lancet last year."

41. Subsequent to the Company's June 11, 2009 release, Medivation held a Biotechnology and Medical Technology Conference at Needham & Company, in which defendant Hung represented the following:

Dimebon works in a very different way than all currently marketed drugs.

* * *

If you look now at this slide here, in addition to preventing neuron death, Dimebon does something else that is quite unusual. This is looking at the amount of sprouts from neurons, so neurons all sprout these connections like roots from a plant, and they have to synapse with each other to make brain connections.

If you look at the far left blue bar, that's the baseline amount of neuron sprouting you'll see in a normal neuron. If you expose any normal neurons to maximal levels of a protein called BDNF – brain-derived neurotrophic factor. This is a protein that is sky-high in developing fetal brains, for instance, because babies' brains grow very, very rapidly. You can see that you get a significant amount of neurite outgrowth.

But to the right of the green bar, if you look at increasing concentrations of Dimebon, Dimebon is a small molecule that causes much neurite outgrowth, has maximally effective concentrations of one of the most potent known growth factors for neurite outgrowth. This is a very unusual finding of the small molecule – can recapitulate the new trophic neutral activity of an andogenous protein. So not only have we shown that we can inhibit neuron death, we are also causing the sprouting of neurons, which we believe are contributing to some of the clinical effects that we are observing.

* * *

Dimebon in the orange is focused on a far different and far more distal target. We are really targeting mitochondrial dysfunction because we believe that all neurodegeneration culminates in mitochondrial dysfunction, which leads to neuron death, synapse loss, and clinical impairment. ***So instead of going for one upstream target like many of the drugs in development, we are going for a downstream target which we think has much broader and more significant effects***.

* * *

The rationale for all of these studies that we are doing is to ultimately increase the commercial value of Dimebon. The broader label we think will help differentiate Dimebon from the generic competition in 2010, when most drugs go generic. We also believe it may facilitate premium pricing, and certainly set the bar higher for our competitors. All of these studies are also further risk mitigation for any single study.

We announced a long time ago that, if we were to do all five pivotals to support the broadest possible label, we would file no later than 2011. But we announced more recently now, especially with this new safety study, that we could file earlier just off the Lancet/CONNECTION and safety studies. So we are kind of keeping both options open.

* * *

[PARTICIPANT:] Then the question on Dimebon, is when would you potentially stop? I mean, I understand when you're going to start but how long would you keep it going?

[HUNG:] So I think what you just raised is a really insightful point. So you know, people have always said that your drug can either be symptomatic or disease-modified, but not both. That is actually totally wrong.

So if you look at rheumatoid arthritis, that is a perfect example of a drug that does both. So if you look at Remicade in rheumatoid arthritis, Remicade improves symptoms immediately. You get effects – a patient will notice effects in days. Yet, if you look over a period of years, there's a clear change in bone loss, and there's clear markers of actual disease modification. So this is a perfect example of a drug that both has symptomatic effects and those disease-modifying effects.

Well, if you looked at our Lancet paper, we are seeing effects with Dimebon at our first time point measured. In fact, at three months – we didn't measure it any earlier than three months but we saw it as early as three months out.

If you look at our preclinical models, in our animals, we see affects [sic] in minutes to hours, and we see effects on cells and the mitochondria in minutes to hours. So we have early near-term effects. We see affects [sic] in preventing neuron death that occur minutes after administration. These cells are much more resistant to things that would otherwise kill them.

But if you don't die in the first minute, you don't die in the next year, I mean, they could still be related processes. One will cause a symptomatic benefits [sic]; the other will cause benefits that will accrue over time. That's what we think we are seeing. We are seeing effects that we – we see short-term effects that we believe are contributing to some of the symptomatic effects that we're seeing. We're also seeing improving differences in patients over time that we believe may actually be disease-

modifying. We're actually doing – our one-year CONCERT study is designed to actually show this increasing effect (inaudible) over time because the EMEA has recently recognized that they will now accept a new label called "delay of disability" for any company that can show with their drug that they increase their clinical effects over a one-year period. So that's what our trial is going for.

So we think that this drug has the potential to be both symptomatic and disease-modifying. That's what we're going for in our label ultimately.

[PARTICIPANT:] When would you stop using the therapy?

[HUNG:] Well, given the fact that Alzheimer's is a constant battle between living cells and dying cells – all of our brains are going through this – so when you are a baby, there's a lot more growth than death; when you're old, there's a lot more death than growth. It's always an equilibrium. Or you're on that equilibrium (inaudible) how big your brain is.

So what we've shown is that Dimebon inhibits the amount of cells that die and may actually cause these sprouts which maybe kind of shift you back to where you are supposed to be. So presumably – we are never going to get rid of all the bad things that happen as you get older; it is overwhelming how many things happen to you in every organ. So you're never going to stop death, so you're going to have to give this drug I think forever. I mean, you can presumably continue to treat people forever and because you are always going to be fighting all of the bad things that happen to you as you age. We intend – the drug is so well-tolerated right now, our safety profile is so attractive that we believe that giving this over a long period of time will not be in issue. In fact, this is one of the easier drugs to take for a long time.

42. On July 12, 2009, Medivation issued a press release entitled "Pfizer and Medivation Present Positive Safety and Tolerability Data on Dimebon in Combination With Donepezil at the International Conference on Alzheimer's Disease," which stated in part:

Pfizer and Medivation, Inc. today announced that new Phase 1 data showed that the investigational drug dimebon (latrepirdine) was well tolerated when used in combination with donepezil HCl tablets, the leading Alzheimer's disease (AD) medication worldwide, in patients with mild-to-moderate Alzheimer's disease. The Phase 1 data were presented today at the 2009 Alzheimer's Association International Conference on Alzheimer's disease (ICAD 2009) in Vienna, Austria.

There were no serious adverse events reported in the study and most adverse events were mild to moderate. The therapy was well tolerated, and all patients completed the treatment period except for one placebo patient. There were no remarkable changes to vital signs, electrocardiograms or laboratory values associated with dimebon treatment.

"Every patient is uniquely affected by Alzheimer's disease and management is often complex. Combination therapy may be needed to maximize clinical benefit, but limited treatment options are available currently," said Pierre N. Tariot, M.D., one of the study's investigators and Director, Memory Disorders Center, Banner Alzheimer's Institute. ***"The Phase 1 data are encouraging, and serve as the foundation for the ongoing Phase 3 CONCERT study, which recently started enrollment in the U.S. and internationally."***

> The CONCERT study is a 12-month clinical trial designed to evaluate the safety and efficacy of dimebon (latrepirdine) when added to ongoing treatment with donepezil in patients with mild-to-moderate AD. CONCERT is designed to complement previous and ongoing studies by further evaluating the efficacy of dimebon and benefits in Alzheimer's disease.

(Footnote omitted.)

43. On August 5, 2009, Medivation reported its second quarter 2009 financial results in a release which stated in part:

> Medivation, Inc. today reported on its corporate progress and financial results for the second quarter ended June 30, 2009.
>
> "In the past several months, we completed enrollment in our confirmatory, pivotal Phase 3 CONNECTION trial of dimebon (latrepirdine) in Alzheimer's disease; initiated the pivotal Phase 3 HORIZON trial of dimebon in Huntington disease; reported important clinical data from both our dimebon and MDV3100 development programs; and raised $62.4 million to further solidify our financial foundation," said David Hung, M.D., president and chief executive officer of Medivation. "We remain focused on executing our milestones, including initiating three additional Phase 3 trials before year end: two dimebon trials in moderate-to-severe Alzheimer's patients and a trial of MDV3100 in advanced prostate cancer."
>
> Recent Accomplishments and Near-Term Milestones
>
> Dimebon (latrepirdine)
>
> - Completed patient enrollment in CONNECTION, a confirmatory, pivotal Phase 3 trial in patients with mild-to-moderate Alzheimer's disease.
> - Continued patient enrollment in the Phase 3 CONCERT trial, a 12-month clinical trial in patients with mild-to-moderate Alzheimer's disease that is designed to evaluate the efficacy of dimebon when added to ongoing treatment with donepezil (Aricept(R)), the leading Alzheimer's disease medication worldwide.
> - Continued enrollment in a placebo-controlled Phase 3 safety study in 750 Alzheimer's disease patients on a variety of background anti-dementia drugs. The purpose of the safety study is to generate a sufficient safety database to provide the option for an earlier-than-planned filing of the initial marketing application should Medivation and Pfizer elect to pursue that option.
> - On track to initiate two additional Phase 3 trials this year that will evaluate dimebon in a total of approximately 1,100 patients with moderate-to-severe Alzheimer's disease.
> - Presented at the Alzheimer's Association 2009 International Conference on Alzheimer's Disease (ICAD) positive safety and tolerability data from a Phase 1 trial showing that dimebon was well tolerated when used in combination with donepezil in patients withmild-to-moderate Alzheimer's disease.

- Initiated HORIZON, a six-month, double-blind, placebo-controlled Phase 3 trial to evaluate dimebon's potential benefits on cognition in patients with Huntington disease.

- Received orphan drug designation for dimebon from the U.S. Food and Drug Administration for the treatment of Huntington disease.

* * *

Corporate

- Successfully raised net proceeds of $62.4 million in a public offering of the Company's common stock.

(Footnote omitted.)

44. On November 3, 2009, Medivation issued a press release entitled "Pfizer And Medivation Initiate Two Phase 3 Trials Of Dimebon In Patients With Moderate-To-Severe Alzheimer's Disease," which stated in part:

> Pfizer Inc and Medivation, Inc. today announced the initiation of CONTACT and CONSTELLATION, two Phase 3 trials of the investigational drug dimebon (latrepirdine) in patients with moderate-to-severe Alzheimer's disease (AD).
>
> The CONTACT study will assess as primary endpoints the potential benefits of adding dimebon to ongoing treatment with donepezil HCl tablets, the leading AD medication worldwide, on neuropsychiatric symptoms and activities of daily living. The CONSTELLATION study will evaluate as primary endpoints the effects of adding dimebon to memantine HCl, another standard of care, on cognition, memory and activities of daily living.
>
> "Alzheimer's disease is a growing global epidemic with an unmet clinical need. Many patients with moderate-to-severe Alzheimer's disease experience behavioral and neuropsychiatric symptoms, which are among the leading causes of placement in care facilities for these patients," said Pierre N. Tariot, MD, director of the Memory Disorders Center at the Banner Alzheimer's Institute and study investigator. "These studies are intended to evaluate the potential added benefits of dimebon in combination with current standards of Alzheimer's care."
>
> ***In preclinical studies, dimebon has been shown to protect brain cells from damage and enhance brain cell survival, potentially by stabilizing and improving mitochondrial function. The dimebon mechanism is distinct from currently available AD medications.***
>
> ***"Pfizer and Medivation are committed to developing dimebon as a treatment that may meaningfully improve the lives of patients across the full spectrum of Alzheimer's disease severity," said Lynn Seely, M.D., chief medical officer for Medivation. "The initiation of the CONTACT and CONSTELLATION studies is an important milestone in the broad clinical development of dimebon."***
>
> These studies are part of a comprehensive Phase 3 clinical development program, currently consisting of seven trials, to assess the safety and efficacy of dimebon across all stages of Alzheimer's disease, as monotherapy and in

combination with currently available Alzheimer's treatments, and in Huntington disease.

(Footnote omitted.)

45. On November 4, 2009, Medivation reported its third quarter 2009 financial results, in a release which stated in part:

> Medivation, Inc. today reported on its corporate progress and financial results for the third quarter ended September 30, 2009.
>
> "With the signing of our agreement for MDV3100 with Astellas last week, we now have a first-class partner with a global reach, leading commercial presence in the urology space, and strategic focus on oncology. This achievement marks our second major collaboration in just over a year's time, bringing us significant resources which allow us to drive our product candidates forward, while still maintaining substantial ownership of our dimebon and MDV3100 programs. We and Astellas are committed to advancing development of this novel androgen receptor antagonist as quickly as possible for a broad spectrum of prostate cancer disease states," said David Hung, M.D., president and chief executive officer of Medivation. ***"We also made important progress with dimebon and now have seven pivotal trials in our broad clinical development program in both Alzheimer's and Huntington diseases in various stages of activity. We have reported results from our first pivotal trial, we expect data in the first half of next year from our second confirmatory pivotal trial, and five other pivotal trials are ongoing."***
>
> *Recent Accomplishments and Near-Term Milestones*
>
> *Dimebon (latrepirdine)*
>
> – On track to announce top-line results from CONNECTION, a confirmatory, pivotal Phase 3 trial in patients with mild-to-moderate Alzheimer's disease, in the first half of 2010.
>
> – Completed patient enrollment in a placebo-controlled Phase 3 safety study in 750 Alzheimer's disease patients on a variety of background anti-dementia drugs.
>
> – Initiated patient enrollment in CONSTELLATION, a six-month, randomized, double-blind, placebo-controlled Phase 3 trial in approximately 570 patients with moderate-to-severe Alzheimer's disease that will evaluate as primary endpoints the effects of adding dimebon to Namenda(R), a standard of care Alzheimer's disease medicine, on cognitive and behavioral symptoms.
>
> – Initiated patient enrollment in CONTACT, a six-month, randomized, double-blind, placebo-controlled Phase 3 trial in approximately 600 patients with moderate-to-severe Alzheimer's disease that will assess as primary endpoints the potential benefits of adding dimebon to ongoing treatment with Aricept(R), the leading Alzheimer's medication worldwide, on neuropsychiatric symptoms and activities of daily living. This study is the first pivotal Alzheimer's disease study to use the Neuropsychiatric Inventory (NPI) scale as a co-primary endpoint.

- Continued patient enrollment in CONCERT, a 12-month Phase 3 clinical trial in patients with mild-to-moderate Alzheimer's disease that is designed to evaluate the efficacy of dimebon when added to ongoing treatment with Aricept.

- Continued patient enrollment in HORIZON, a six-month, double-blind, placebo-controlled Phase 3 trial that is evaluating dimebon's potential benefits on cognition in patients with Huntington disease.

(Footnote omitted.)

46. On February 19, 2010, defendant Machado sold 30,000 shares of his Medivation stock at $37.26 per share for gross proceeds of $1.1 million.

47. On March 2, 2010, Medivation's stock closed at $40.25 per share, its Class Period and all-time high.

48. Then, on March 3, 2010, before the market opened, Medivation issued a press release entitled "Pfizer And Medivation Announce Results From Two Phase 3 Studies In Dimebon (latrepirdine) Alzheimer's Disease Clinical Development Program," which stated in part:

> Pfizer Inc. and Medivation, Inc. today announced results from two Phase 3 trials of the investigational drug dimebon (latrepirdine) in patients with Alzheimer's disease (AD). ***In the CONNECTION trial, dimebon did not meet its co-primary or secondary efficacy endpoints compared to placebo. Co-primary endpoints were measures of cognition and global function.***
>
> ***"The results from the CONNECTION study are unexpected, and we are disappointed for the Alzheimer's community,"*** said Dr. David Hung, president and chief executive officer of Medivation. "We are working with our colleagues at Pfizer to better understand the CONNECTION data and we plan to present these data at an upcoming medical meeting."
>
> Dimebon was well tolerated in both the CONNECTION study and in a separate Phase 3 safety and tolerability study, which confirmed dimebon's tolerability when dosed alone or in combination with approved Alzheimer's disease medicines.
>
> "We are evaluating the CONNECTION data with Medivation. After that review, Pfizer will be in a position to determine appropriate next steps regarding the dimebon program," said Dr. Briggs W. Morrison, senior vice president, clinical development, Primary Care Business Unit at Pfizer. "We recognize the significant medical need, and we are committed to advancing treatment options for Alzheimer's disease."

(Footnote omitted.)

49. On this news, Medivation's shares plummeted $27.15 per share from their Class Period high of $40.25 per share to close at $13.10 per share on March 3, 2010 – a one-day decline of 67% on volume of 45 million shares, following the announcement.

50. On March 3, 2010, *The Science Business* published a healthcare blog entitled "Medivation Alzheimer's Drug Was Hyped," which stated in part:

> Shares of Medivation plunged 67% today after the Alzheimer's drug it was developing with Pfizer failed abysmally in its first big clinical trial. Investors and some Alzheimer's researchers had had high hope that the drug, called Dimebon, would be the first drug to slow the course of the disease.
>
> But a top doctor from University of Southern California says that there were signs all along that the drug wasn't all it was made out to be.The drug, a former antihistamine sold in Russia, emerged from nowhere a few years ago to become one of the hottest new Alzheimer's drugs in testing. The excitement, however, was based virtually entirely on one smallish trial of under 200 patients conducted in Russia. And the mechanism of action of the drug was murky all along.
>
> "***This drug was so hyped,***" says USC psychiatrist and Alzheimer's expert Lon Schneider "***When you look at this drug [chemically] there is nothing particularly special about it.***" He says its tricyclic chemical structure is roughly similar to lots of antihistamines, antidepressants, and antipsychotic drugs. There is nothing in its structure to indicate it would have remarkable effect, he argues.
>
> Schneider says he has no problem with Pfizer's business decision to gamble on an unproven drug from Russia. What bothers him, he says, is the way Medivation and its allies positioned Dimebon as the next big thing in Alzheimer' disease without good evidence to support this.
>
> ***Medivation has argued for years that there is something unusual about the drug. It has pointed to lab evidence and suggests that the drug [sic] not merely a symptom enhancer, but might actually slow the course of the disease over time. In particular, the company has pushed the concept that the main effect of the drug is to boost the health of energy producing structures inside cells called mitochondria.***
>
> Some evidence definitely supports the idea that Dimebon hits mitochondria. But Schneider says that lab data also shows the drug hits all sorts of other brain chemicals including serotonin and dopamine. Emphasizing mitochodria, he says "is just cherry-picking a particular mechanism of action that may or may not be relevant."
>
> Schneider points to an independent study showing that while the drug can have neuroprotective effects in animals, the concentrations of the drug achieved in humans are far to low [sic] to have neuroprotective effects. The 2009 study from the UT Southwestern Medical Center in Dallas, published in the journal Molecular Neurodegeneration, concluded that the high concentration of Dimebon required to achieve neuroprotective effects in animals "is not likely to be achieved in human trials." (Schneider has consulted for Pfizer, Medivation, and other companies testing Alzheimer's drugs.)

51. Defendants violated Rule 10b-5 by misrepresenting, obfuscating, and concealing critical information about Dimebon so as to keep the public from obtaining a meaningful understanding of the drug's prospects and market success.

52. As a result of defendants' false statements, Medivation's stock traded at inflated levels during the Class Period. However, after the above revelations seeped into the market, the Company's shares were hammered by massive sales, sending them down more than 67% from their Class Period high.

LOSS CAUSATION/ECONOMIC LOSS

53. During the Class Period, as detailed herein, defendants engaged in a scheme to deceive the market and a course of conduct that artificially inflated Medivation's stock price and operated as a fraud or deceit on Class Period purchasers of Medivation stock by misrepresenting the Company's key product and the implications of the findings from earlier studies on Dimebon. Later, however, when defendants' prior misrepresentations and fraudulent conduct were disclosed and became apparent to the market, Medivation stock fell precipitously as the prior artificial inflation came out of Medivation's stock price. As a result of their purchases of Medivation stock during the Class Period, plaintiff and other members of the Class suffered economic loss, *i.e.*, damages under the federal securities laws.

54. Defendants' false and misleading statements had the intended effect and caused Medivation stock to trade at artificially inflated levels throughout the Class Period, reaching as high as $40.25 per share.

55. On March 3, 2010, before the market opened, defendants were forced to publicly disclose that Dimebon had failed its first late phase clinical trial as the drug did not meet its primary or secondary endpoints. These public revelations indicated that the prior representations about Dimebon for treatment of Alzheimer's disease had been false. As investors and the market became aware that Medivation's statements had been false and misleading and that Medivation's actual business prospects, which had long been obfuscated by the scheme to distort the study results, were, in fact, poor, the prior artificial inflation came out of Medivation's stock price, damaging investors.

56. As a direct result of defendants' admissions and the public revelations regarding the truth about Medivation's key drug and its actual business prospects going forward, Medivation's stock price plummeted 67%, on unusually high volume, falling from $40.25 on March 2, 2009, to $13.10 per share on March 3, 2010. This drop removed the inflation from Medivation's stock price, causing real economic loss to investors who had purchased the stock during the Class Period.

57. The 67% decline in Medivation's stock price at the end of the Class Period was a direct result of the nature and extent of defendants' fraud finally being revealed to investors and the market. The timing and magnitude of Medivation's stock price decline negates any inference that the loss suffered by plaintiff and other Class members was caused by changed market conditions, macroeconomic or industry factors or Company-specific facts unrelated to the defendants' fraudulent conduct. During the same period in which Medivation's stock fell 67% from $40.25 per share as a result of defendants' fraud being revealed, the Standard & Poor's 500 securities index was flat. The economic loss, *i.e.*, damages, suffered by plaintiff and other members of the Class, was a direct result of defendants' fraudulent scheme to artificially inflate Medivation's stock price and the subsequent significant decline in the value of Medivation's stock when defendants' prior misrepresentations and other fraudulent conduct was revealed.

COUNT I

For Violation of §10(b) of the 1934 Act and Rule 10b-5 Against All Defendants

58. Plaintiff incorporates ¶¶1-57 by reference.

59. During the Class Period, defendants disseminated or approved the false statements specified above, which they knew or deliberately disregarded were misleading in that they contained misrepresentations and failed to disclose material facts necessary in order to make the statements made, in light of the circumstances under which they were made, not misleading.

60. Defendants violated §10(b) of the 1934 Act and Rule 10b-5 in that they:

(a) Employed devices, schemes, and artifices to defraud;

(b) Made untrue statements of material facts or omitted to state material facts necessary in order to make the statements made, in light of the circumstances under which they were made, not misleading; or

(c) Engaged in acts, practices, and a course of business that operated as a fraud or deceit upon plaintiff and others similarly situated in connection with their purchases of Medivation common stock during the Class Period.

61. Plaintiff and the Class have suffered damages in that, in reliance on the integrity of the market, they paid artificially inflated prices for Medivation common stock. Plaintiff and the Class would not have purchased Medivation common stock at the prices they paid, or at all, if they had been aware that the market prices had been artificially and falsely inflated by defendants' misleading statements.

62. As a direct and proximate result of these defendants' wrongful conduct, plaintiff and the other members of the Class suffered damages in connection with their purchases of Medivation common stock during the Class Period.

COUNT II

For Violation of §20(a) of the 1934 Act Against All Defendants

63. Plaintiff incorporates ¶¶1-62 by reference.

64. The Individual Defendants acted as controlling persons of Medivation within the meaning of §20(a) of the 1934 Act. By reason of their positions as officers and/or directors of Medivation and their ownership of Medivation stock, the Individual Defendants had the power and authority to cause Medivation to engage in the wrongful conduct complained of herein. Medivation controlled each of the Individual Defendants and all of its employees. By reason of such conduct, the Individual Defendants and Medivation are liable pursuant to §20(a) of the 1934 Act.

PRAYER FOR RELIEF

WHEREFORE, plaintiff prays for judgment as follows:

A. Declaring this action to be a proper class action pursuant to Fed. R. Civ. P. 23;

B. Awarding plaintiff and the members of the Class damages, including interest;

C. Awarding plaintiff reasonable costs and attorneys' fees; and

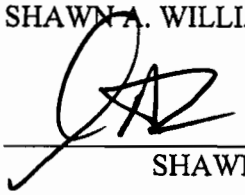
D. Awarding such equitable/injunctive or other relief as the Court may deem just and proper.

JURY DEMAND

Plaintiff demands a trial by jury.

DATED: March 9, 2010

COUGHLIN STOIA GELLER
RUDMAN & ROBBINS LLP
SHAWN A. WILLIAMS



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CERTIFICATION OF INTERESTED ENTITIES OR PERSONS

Pursuant to Civil L.R. 3-16, the undersigned certifies that as of this date, other than the named parties, there is no such interest to report.

ATTORNEY OF RECORD FOR PLAINTIFF
DAVID APPLESTEIN

Plaintiff's Certification

I, David Applestein, do hereby certify that:

1. I have reviewed the complaint and authorize its filing.

2. I purchased the securities of Medivation Inc. which are the subject of the complaint, but not at the direction of my counsel or in order to participate in any private action under the Securities Act of 1933 or Securities Exchange Act of 1934.

3. I am willing to serve as a representative party on behalf of a class, including providing testimony at deposition and trial if necessary.

4. During the three year period prior to the date of this certification, I have not sought to serve as a representative party on behalf of a class in an action brought under the federal securities laws.

5. During the Class Period, July 17, 2008 through March 2, 2010, inclusive, I engaged in the following transactions in Medivation Inc.:

Buy/Sell	Trade date	No. of securities	Price
buy	7/31/08	300	$19.94 per share

6. I will not accept any payment for serving as a representative party on behalf of the class beyond my pro rata share of any recovery, except such reasonable costs and expenses (including lost wages) directly related to the representation of the Class and my activities in the lawsuit, as ordered or approved by the Court.

7. Nothing herein shall be construed to be or constitute a waiver of my attorney client privilege.

8. I certify under penalty of perjury that the foregoing is true and correct.

Executed on March 7, 2010

David Applestein

signature